What is claimed is:

1. A method to protect a mammal from a disease characterized by eosinophilia associated with an inflammatory response, said method comprising administering a heat shock protein to a mammal having said disease.

2. The method of Claim 1, wherein said disease is associated with increased production of a cytokine selected from the group consisting of interleukin-4 (IL-4), interleukin-5 (IL-5), interleukin-6 (IL-6), interleukin-9 (IL-9), interleukin-10 (IL-10), interleukin-13 (IL-13) and interleukin-15 (IL-15).

3. The method of Claim 1, wherein said disease is selected from the group consisting of allergic airway diseases, hyper-eosinophilic syndrome, helminthic parasitic infection, allergic rhinitis, allergic conjunctivitis, dermatitis, eczema, contact dermatitis, and food allergy.

4. The method of Claim 1, wherein said disease is a respiratory disease characterized by eosinophilic airway inflammation and airway hyperresponsiveness.

5. The method of Claim 4, wherein said respiratory disease is selected from the group consisting of allergic asthma, intrinsic asthma, allergic bronchopulmonary aspergillosis, eosinophilic pneumonia, allergic bronchitis bronchiectasis, occupational asthma, reactive airway disease

syndrome, interstitial lung disease, hyper-eosinophilic syndrome, and parasitic lung disease.

6. The method of Claim 1, wherein said disease is associated with sensitization to an allergen.

7. The method of Claim 1, wherein said disease is allergic asthma.

8. The method of Claim 1, wherein said heat shock protein is selected from the group consisting of an HSP-60 family heat shock protein, an HSP-70 family heat shock protein, an HSP-90 family heat shock protein and an HSP-27 family heat shock protein.

9. The method of Claim 1, wherein said heat shock protein is selected from the group consisting of an HSP-60 family heat shock protein, an HSP-70 family heat shock protein and an HSP-27 family heat shock protein.

10. The method of Claim 1, wherein said heat shock protein is selected from the group consisting of an HSP-90 family heat shock protein and an HSP-27 family heat shock protein.

11. The method of Claim 1, wherein said heat shock protein is selected from the group consisting of a bacterial heat shock protein and a mammalian heat shock protein.

12. The method of Claim 1, wherein said heat shock protein is a mycobacterial heat shock protein.

13. The method of Claim 1, wherein said heat shock protein is a mycobacterial heat shock protein-65 (HSP-65).

14. The method of Claim 1, wherein said heat shock protein is administered by at least one route selected from the group consisting of oral, nasal, topical, inhaled, transdermal, rectal and parenteral routes.

15. The method of Claim 1, wherein said heat shock protein is administered by a route selected from the group consisting of inhaled and nasal routes.

16. The method of Claim 1, wherein said heat shock protein reduces eosinophilia in said mammal.

17. The method of Claim 1, wherein said heat shock protein reduces eosinophil blood counts in said mammal to between about 0 and about 300 cells/mm^3.

18. The method of Claim 1, wherein said heat shock protein reduces eosinophil blood counts in said mammal to between about 0 and about 100 cells/mm^3.

19. The method of Claim 1, wherein said heat shock protein reduces eosinophil blood counts in said mammal to between about 0% and about 3% of total white blood cells in said mammal.

20. The method of Claim 1, wherein said heat shock protein induces interferon-γ (IFN-γ) production by T lymphocytes in said mammal.

21. The method of Claim 1, wherein said heat shock protein suppresses interleukin-4 (IL-4) and interleukin-5 (IL-5) production by T lymphocytes in said mammal.

22. The method of Claim 1, wherein said heat shock protein decreases airway methacholine responsiveness in said mammal.

23. The method of Claim 1, wherein said heat shock protein reduces airflow limitation in said mammal such that an FEV_1/FVC value of said mammal is at least about 80%.

24. The method of Claim 1, wherein said heat shock protein results in an improvement in a mammal's $PC_{20methacholine}FEV_1$ value such that the $PC_{20methacholine}FEV_1$ value obtained before administration of said heat shock protein when the mammal is provoked with a first concentration of methacholine is the same as the $PC_{20methacholine}FEV_1$ value obtained after administration of said heat shock protein when the mammal is provoked with double the amount of the first concentration of methacholine.

25. The method of Claim 24, wherein said first concentration of methacholine is between about 0.01 mg/ml and about 8 mg/ml.

26. The method of Claim 1, wherein said heat shock protein improves a mammal's FEV_1 by between about 5% and about 100% of said mammal's predicted FEV_1.

27. The method of Claim 1, wherein said heat shock protein reduces airflow limitation in said mammal such that an R_L value of said mammal is reduced by at least about 20%.

28. The method of Claim 1, wherein said heat shock protein is administered in an amount between about 0.1 microgram x kilogram^{-1} and about 10 milligram x kilogram^{-1} body weight of a mammal.

29. The method of Claim 1, wherein said heat shock protein is administered in an amount between about 1 microgram x kilogram^{-1} and about 1 milligram x kilogram^{-1} body weight of a mammal.

30. The method of Claim 1, wherein said heat shock protein is administered in an amount between about 0.1 milligram x kilogram^{-1} and about 5 milligram x kilogram^{-1} body weight of a mammal, if said heat shock protein is delivered by aerosol.

31. The method of Claim 1, wherein said heat shock protein is administered in an amount between about 0.1 microgram x kilogram^{-1} and about 10 microgram x kilogram^{-1} body weight of a mammal, if said heat shock protein is delivered parenterally.

32. The method of Claim 1, wherein said heat shock protein is administered in a pharmaceutically acceptable excipient.

33. The method of Claim 1, wherein said mammal is a human.

34. A method for prescribing treatment for airway hyperresponsiveness or airflow limitation associated with a disease involving an inflammatory response, comprising:

a. administering to a mammal a heat shock protein;

b. measuring a change in lung function in response to a provoking agent in said mammal to determine if said heat shock protein modulates airway hyperresponsiveness or airflow limitation; and,

c. prescribing a pharmacological therapy comprising administration of said heat shock protein to said mammal effective to reduce inflammation based upon said changes in lung function.

35. The method of Claim 34, wherein said disease is characterized by airway eosinophilia.

36. The method of Claim 34, wherein said provoking agent is selected from the group consisting of a direct and an indirect stimuli.

37. The method of Claim 34, wherein said provoking agent is selected from the group consisting of an allergen, methacholine, a histamine, a leukotriene, saline, hyperventilation, exercise, sulfur dioxide, adenosine,

propranolol, cold air, an antigen, bradykinin, acetylcholine, a prostaglandin, ozone, environmental air pollutants and mixtures thereof.

38. The method of Claim 34, wherein said step of measuring comprises measuring a value selected from the group consisting of FEV_1, FEV_1/FVC, $PC_{20methacholine}FEV_1$, post-enhanced h (Penh), conductance, dynamic compliance, lung resistance (R_L), airway pressure time index (APTI), and peak flow.

39. A method to protect a mammal from a disease characterized by airway hyperresponsiveness associated with an inflammatory response, said method comprising administering a heat shock protein to a mammal having said disease.

40. A method to protect a mammal from an inflammatory disease characterized by a Th2-type immune response, said method comprising administering a heat shock protein to a mammal having said disease.

41. A formulation for protecting a mammal from developing a disease characterized by eosinophilia associated with an inflammatory response, comprising a heat shock protein and an anti-inflammatory agent.

42. The formulation of Claim 41, wherein said anti-inflammatory agent is selected from the group consisting of an antigen, an allergen, a hapten, proinflammatory cytokine antagonists, proinflammatory cytokine receptor antagonists, anti-CD23, anti-IgE, leukotriene synthesis inhibitors, leukotriene receptor antagonists, glucocorticosteroids, steroid chemical derivatives, anti-cyclooxygenase agents, anti-cholinergic agents, beta-adrenergic agonists, methylxanthines, anti-histamines, cromones, zyleuton, anti-CD4 reagents, anti-IL-5 reagents, surfactants, anti-thromboxane reagents, anti-serotonin reagents, ketotiphen, cytoxin, cyclosporin, methotrexate, macrolide antibiotics, heparin, low molecular weight heparin, and mixtures thereof.

43. The formulation of Claim 41, wherein said formulation comprises a pharmaceutically acceptable excipient.

44. The formulation of Claim 41, wherein said formulation comprises a pharmaceutically acceptable excipient selected from the group consisting of biocompatible polymers, other polymeric matrices, capsules, microcapsules, microparticles, bolus preparations, osmotic pumps, diffusion

devices, liposomes, lipospheres, and transdermal delivery systems.

45. The method of Claim 41, wherein said heat shock protein is selected from the group consisting of an HSP-60 family heat shock protein, an HSP-70 family heat shock protein, an HSP-90 family heat shock protein and an HSP-27 family heat shock protein.

46. The method of Claim 41, wherein said heat shock protein is a mycobacterial heat shock protein.

47. The method of Claim 41, wherein said heat shock protein is a mycobacterial heat shock protein-65 (HSP-65).

48. A method to protect a mammal from a disease identified by a characteristic selected from the group consisting of eosinophilia, airway hyperresponsiveness and a Th2-type immune response, said characteristic being associated with an inflammatory response, said method comprising administering a nucleic acid molecule encoding a heat shock protein to a mammal having said disease.

49. The method of Claim 48, wherein said nucleic acid molecule is operatively linked to a transcription control sequence.

50. The method of Claim 48, wherein said nucleic acid molecule is administered with a pharmaceutically acceptable excipient selected from the group consisting of an aqueous physiologically balanced solution, an artificial lipid-containing substrate, a natural lipid-containing substrate, an oil, an ester, a glycol, a virus, a metal particle and a cationic molecule.

51. The method of Claim 48, wherein said pharmaceutically acceptable excipient is selected from the group consisting of liposomes, micelles, cells and cellular membranes.

52. The method of Claim 48, wherein said nucleic acid molecule is administered by a mode selected from the group consisting of intradermal injection, intramuscular injection,

intravenous injection, subcutaneous injection, and *ex vivo* administration.